

Revisiting the Syndrome of “Obsessional Slowness”

Christos Ganos, MD,^{1,2} Panagiotis Kassavetis, MD,¹ Maria Cerdan, MD,^{1,3} Roberto Erro, MD,¹ Bettina Balint, MD,¹ Gary Price, MD,⁴ Mark J. Edwards, MD, PhD,¹ Kailash P. Bhatia, MD^{1,*}

Abstract: Background: Obsessional slowness (OS) denotes a rare condition of disablingly slow motor performance. It was originally described in patients with obsessive-compulsive disorder as a “primary” condition; however, subsequent reports have included heterogeneous clinical populations. We wished to reassess patients with this diagnosis at our own institution and also revisit the literature to provide an overview of this condition.

Methods: Clinical documentation and videos of 3 patients diagnosed with OS in the National Hospital for Neurology and Neurosurgery (London, UK) were reviewed. One of the patients was clinically reappraised. A systematic review of published articles with sufficient clinical patient information was also conducted.

Results: Our 3 cases were male with symptom onset in adolescence or early adulthood. Motor slowness with poverty of movement and a history of obsessive-compulsive symptoms were characteristic. Poor speech production, bizarre postures, mannerisms, echophenomena, and oculogyric tics were also noted. Dopaminergic imaging was normal in 2 cases. One case had autistic features. Systematic literature review identified 77 further cases. Male preponderance with symptom onset mainly during the second decade and presence of obsessive-compulsive symptoms were noted. Additional motor and neuropsychiatric features were often present.

Conclusion: The existence of OS as a “primary” condition is doubtful. This diagnosis has been given to characterize different clinical presentations ranging from obsessive-compulsive disorder with motor slowness resulting from covert obsessive-compulsive symptoms to catatonia. Clinicians should be aware of this syndrome to separate it from juvenile parkinsonism and other causes of motor slowness given that diagnostic approaches and treatment strategies differ.

An extremely rare condition termed obsessional slowness (OS) has been described in the medical literature, with the first report published in 1974.¹ It has been defined as a clinical syndrome where patients are very slow in motor performance. It was proposed to be a primary phenomenon, though the original description was in a cohort of patients with obsessive-compulsive disorder (OCD). The proposal was that though patients with OS did have OCD, the motor symptoms were not related to the presence of obsessions or compulsions that might be expected to cause motor slowness (such as checking and mental rituals), and instead it was a separate and “primary” phenomenon.

It was described as a cause of significant disruption in the execution of personal and social activity to the level of incapacitation. However, since the earliest reports, controversy has existed as to whether OS is truly a distinct and primary entity or may, on grounds of phenomenology, be the expression of a range of other conditions.^{2–4}

Given the extreme rarity of this diagnosis and its debated nature as a distinct clinical syndrome, we carefully examined characteristics of 3 patients who had previously received the diagnosis of OS in our clinic after extensive neurological and psychiatric assessment and investigations and took the opportu-

¹Sobell Department of Motor Neuroscience and Movement Disorders, UCL Institute of Neurology, University College London, London, United Kingdom; ²Department of Neurology, University Medical Center Hamburg-Eppendorf (UKE), Hamburg, Germany; ³Department of Neurology, Hospital Universitario Virgen de La Arrixaca, Murcia, Spain; ⁴Department of Neuropsychiatry, The National Hospital for Neurology and Neurosurgery, London, United Kingdom

*Correspondence to: Prof. Kailash P. Bhatia, Sobell Department of Motor Neuroscience and Movement Disorders, UCL Institute of Neurology, Queen Square, London, WC1N 3BG, United Kingdom; E-mail: K.bhatia@ion.ucl.ac.uk

Keywords: obsessional slowness, catatonia, obsessive-compulsive disorder, motor slowness.

Relevant disclosures and conflicts of interest are listed at the end of this article.

Received 22 September 2014; revised 10 December 2014; accepted 10 December 2014.

Published online 16 March 2015 in Wiley InterScience (www.interscience.wiley.com). DOI:10.1002/mdc3.12140

nity to systematically review the literature in order to be able to best place these cases within the spectrum of disorders that may be diagnosed with OS.

Patients and Methods

From the total cohort of patients who attended the movement disorders clinic of the National Hospital for Neurology and Neurosurgery (London, UK), we identified 3 patients diagnosed with OS. Each patient was jointly diagnosed by a neuropsychiatrist and a neurologist. Clinical records, including video recordings, were reviewed in these cases. One patient (case 1) was also clinically reappraised. Patient-written informed consents for video recordings and publication were obtained according to local standard protocols and the Data Protection Act 1998.

Furthermore, a Medline search (by PubMed, a service of the National Library of Medicine's National Center for Biotechnology Information; <http://www.ncbi.nlm.nih.gov>) for all publications in the English language with the terms of “obsessional/obsessive slowness” was conducted and articles were screened for the presence of OS cases. Abstracts of references of identified publications were also screened for the presence of additional cases. Only articles that provided sufficient clinical information were included.

Clinical Cases

Case 1

At the age of 12, this 22-year-old man, who had normal developmental milestones and social skills, became increasingly obsessive with eating and dressing habits as well as hygiene. He developed excessive repetitive and time-consuming hand-washing rituals and meticulously placed bathroom utensils in order and became fascinated with action figures, which he would obsessively collect. He increasingly isolated himself, and during that period, secondary enuresis was noticed. At age 14, he witnessed a violent event, upon which he “froze,” maintaining the same position with his jaw open for 10 minutes. Over the ensuing years, he became more withdrawn and at age 16 it was noted that his movements were increasingly slow. His speech production was progressively reduced. He had poor appetite, which led to weight loss. He had difficulties with concentration; however, he managed to attend college, but did not complete his curriculum mainly owing to slowness. Given his slowness and other symptoms, he was diagnosed with depression at age 17 and was treated with mirtazapine (45 mg/day) and subsequently olanzapine (15 mg/day) without consistent beneficial effect. The latter was discontinued within 4 weeks because of involuntary arm shaking and jerking, which improved shortly on stopping the medication. He avoided eye contact and was noted to speak to himself and stare at objects. He developed abnormal postures and bizarre movements. He was, however, able to use his computer and felt that he could move normally when left alone. There was no relevant family

history. A trial of lorazepam (5 mg/day) over several weeks at age 19 did not improve his symptoms.

Currently, he is reported to have become “100 times slower.” He barely engages in conversations, but often quietly repeats what is said in his presence (“ambient echolalia”). However, if agitated, he can suddenly “snap” and “become normal again.” He does not take care of himself and has to be instructed to use the bathroom to void his bladder, but has no problems in controlling his bowels. He spends a lot of time in front of the computer. He is on venlafaxine (225 mg/day) and this, paralleled by physiotherapy, has led to some improvement. Although he is unable to report why, he acknowledges that his movements are extremely slow, but denies having anxiety regarding moving more quickly. No psychotic symptoms have presented over time. He is attending a specialist college.

Clinical examination showed normal smooth pursuit and saccadic eye movements, however in the presence of profound distractibility with gaze impersistence and oculogyric tics. Blink rate was normal. There was facial impassivity. Repetitive and stereotypic perioral movements were noted. Speech was markedly reduced, hesitant, and whispering. There was poverty of movement with profoundly prolonged movement initiation, very slow movement execution, and movement interruptions. Muscle tone was normal. There were no sensory and no cerebellar signs. Gait was hesitant and slow. During examination, there were echophenomena, including immediate and delayed echopraxia and echolalia and also ambient echolalia. There was also occasional grimacing, posturing, unusual mannerisms, as well as features of catalepsy and waxy flexibility. Segments of neurological examination are shown in Video 1.

Neuropsychological examination was impaired by his profound slowness. However, the summary was that, on nonverbal tests of intellectual functioning, he was at a modest level.

Extensive investigations were unrevealing (laboratory: serum copper/seruloplasmin, thyroid function, white cell enzymes, classical anti-onconeural/anti-N-methyl-D-aspartate/anti-glutamic acid decarboxylase autoantibodies, acanthocytes, organic amino-acids in plasma and organic acids in urine, cerebrospinal fluid [CSF], and cultured skin fibroblasts for Filipin staining; neurophysiological: electroretinogram, EEG, nerve conduction studies, and brain-stem-evoked potentials; neuroimaging: repeated cranial MRIs, DaTSCAN, 18-fluorodeoxyglucose-PET).

Case 2

This 34-year-old man presented with an 8-year history of insidiously progressive motor slowness. He was first noticed to be slow and hesitant to reply to questions. His walking decelerated and subsequently all his actions became slower. It took him approximately 25 minutes to dress. He did not complain regarding his motor performance. Following a physical assault, his speech became quieter, slurred, and incomprehensible. After alcohol intake or when angry, he would speed up dramatically for short periods of time. Paradoxically, he was a regular swimmer given that swimming was unaffected.

He was born with congenital deafness and had speech and learning difficulties early in life. However, he managed to obtain a satisfactory job in an assembly plant, which he had to give up 2 years after symptom onset. He was particularly meticulous and took care of positioning objects in certain ways and order. There was no history of psychotic symptoms and no family history of neuropsychiatric illness.

On clinical examination (see Video 2), mild dysmorphic features, facial impassivity, lack of spontaneous movements, start hesitation, positive glabellar tap sign, and monotonous and repetitive speech were striking features. There was no rigidity, but paratonia. Movements were slow and, on occasion, manneristic, but there was no fatiguing. Sensory examination was unrevealing. His gait was slow, but with normal stride length. His body posture was stooped and the arms extended away from the body. He had a thorough psychiatric assessment, which did not reveal gross cognitive deficits. However, a formal neuropsychological evaluation was not performed.

Clinical investigations were unrevealing (laboratory: serum copper/ceruloplasmin and thyroid function, acanthocytes, autoantibodies, and anti-cardiolipine; neurophysiological: EEG; neuroimaging: cranial MRI and [^{18}F]dopa-PET).

Case 3

This 29-year-old man presented with a history of chronic extreme motor slowness, which developed at the age of 17. He had initially become socially withdrawn and had stopped verbal communication. He had also become neglectful of himself. He did not feel disturbed by his slowness, even though he needed more than 1 hour to get dressed or get through a meal. He was incontinent at times, as if he could not move to take himself to the toilet, and he also developed unusual mannerisms, but no psychotic symptoms. However, the whole tenor of the illness was dominated by his dramatic slowness of movement and thought.

Although his motor development was normal, there were prominent autistic features. He had been considered a shy “unemotional” child and was always anxious at meeting strangers. He never made any friends, never had any pretend play or interest in imaginative pursuits or stories, and preferred solitude. From the time he was physically able, he was very meticulous with his possessions and made sure they were kept neatly and remained ordered. His only interests from a young age were astronomy and music, and he dropped out school at 15. He could not maintain any job, given that he had to be told what to do and would just do nothing if left to get on by himself.

Clinical investigations were unremarkable (laboratory: serum copper/ceruloplasmin and thyroid function and CSF; neurophysiological: motor- and somatosensory-evoked potentials, EEG, and neuroimaging: cranial MRI). Minimal and only short-lived responses to levodopa, neuroleptic medication, and 3 courses of electroconvulsive therapy were reported. There was no relevant family history.

On examination, there was marked start hesitation for any motor command. There were dystonic facial movements and

fluttering of the eyelids. Marked jaw opening and a delay of approximately 30 seconds would precede usually monosyllabic articulations. Limb movements were performed with extreme slowness. There was no rigidity or tremor. Power and reflexes were normal. His gait appeared awkward and clumsy with his arms held abducted (see Video 3). Postural reflexes were preserved. There was no obvious sensory deficit or cerebellar dysfunction. Ritualistic movements and stereotypies were present.

Review of Literature

Of a total of 58 articles using the terms obsessive/obsessional slowness, 15 with sufficient clinical information for 77 patients were identified.^{1–15} Fifty-nine (76.6%) of the reviewed cases were male. Onset was during the second decade for most cases. OS occurred, in most cases, with some obsessive-compulsive symptoms. Approximately half of the reported cases had additional diagnoses, including Gilles de la Tourette syndrome (GTS), depression, parkinsonism and Down's syndrome.^{2,7,8,10–12,14} In some cases, prominent speech reduction or mutism, incontinence, perseverations, and mannerisms were also noted.^{2,7–10,13} Follow-up information upon diagnosis was provided in 14 cases for a period ranging between 4 weeks and 14 years.^{1,5,6,8–10,12–15} Some clinical improvement was reported in these patients, though OS is a chronic condition and clinical descriptions were, in general, limited (see Table 1 for details).

Discussion

The precise definition of OS is variable within the medical literature. It was originally described as a “primary” condition, but, confusingly, was described in patients with clear diagnosis of OCD.¹ The original description sought to separate the motor phenomena of OS from those that were simply a secondary consequence of typical overt obsessive-compulsive/ritualistic behaviors, such as checking or simple anxiety.¹ From a movement disorders perspective, it was unclear whether slowness was a consequence of movement initiation difficulties, overt slowness of movement execution, or whether was a result of (covert) repetitive compulsive behaviors causing action interruptions and/or disabling distractibility. Subsequent literature has contributed to the terminological diversity of OS by applying different criteria to heterogeneous populations. For example, although, in the first 10 patients reported by Rachman in 1974, slowness was not the result of overt obsessive-compulsive symptoms, such as checking,¹ two subsequent reports used the term OS to describe patients with incapacitating slowness related to mental checking.^{5,6} A subsequent large-scale study, which screened for symptoms of OS in clinical records of 665 cases of OCD patients, applied even more liberal criteria: “slowness not clearly due to washing or fears of contamination.”³ To complicate matters further, Hymas et al. reported on the characteristics of adults with symptoms of OS who underwent behavioral therapies for OCD as inpatients, including those with comorbid GTS or organic mental disorder, highlighting the presence of

TABLE 1 Literature review of cases with OS

First Author	Year	No. of Cases	Gender (M/F)	Age at Onset (Years)	Main Features	Difficulty in Movement Initiation	Slowness of Movement Execution	Checking Behavior/Action Interruptions	Social Impairment	Early Development	Clinical Investigations	Neuroimaging	Tx	Tx Response	Follow-up
Rachmann	1974	10	8/2 ^a	Early adulthood	Incapacitating slowness, mental checking	NA	NA	No prominent checking	Yes	NA	NA	NA	Prompting/pacing/shaping	±	NA ^b
Blisbury and Morley	1979	1	M	20	Slowness, mental checking	None	None	Yes	Yes	Normal	NA	NA	Prompting/pacing	±	19 weeks
Bennun	1980	1	M	19	Slowness, mental checking	None	None	Yes	Yes	Normal	NA	NA	Pacing/self-instruction	+	27 weeks
Clark	1982	1	M	8	Slowness, incontinence, remain motionless, tics, mannerisms, perseverations, OCS	Yes	None	Yes	Yes ('social isolate since preschool years')	Normal	NA	NA	Modeling/prompting/pacing	±	4 weeks
Ramasuriya	1991	22	19/2	19	Slowness, rituals, ruminations	NA	NA	Yes	NA	Prenatal problems = 6, abnormal development = 2	NA	NA	NA	NA	NA
Sawle	1991	6 ^c	6/0	Late adolescence/early adulthood	Slowness, OCS, 1 × depression	Yes	Yes	Yes	NA	NA	NA	¹⁵ Oxygen PET: hypermetabolism at OFC, MFC, and PMC; ¹⁸ F-dopa PET: normal	TCA, neuroleptics, BZP	NA	NA
Hymas	1991	17 ^c	10/7	NA	Slowness, OCS, neurological signs, GTS, parkinsonism, speech abnormalities, depression, perseverations, distractibility	Yes	Yes	Yes	1-ASD	1-abnormal	NA	NA	Neuroleptics, BZP, TCA	NA	NA
Veale and Sawle	1993	3	2/1	Adolescence	Slowness, OCD	Yes	Yes	Yes	NA	NA	NA	NA	NA	NA	NA
	1993	1	M	35	GTS, OCD, slowness	Yes	No	No	Yes	GTS since age 7	NA	¹⁵ Oxygen PET: caudate and thalamic hypermetabolism	Thermocoagulation of anterior cingulum	NA ++	21 months
Tham	1994	1	M	19	Slowness, OCD with fears of uncleanliness and contamination	Yes	NA	Yes	Yes	NA	NA	NA	Prompting/pacing	+	16 months
Takeuchi	1997	4	1/3	Childhood/adolescence	Slowness, OCS/OCD, depression	NA	NA	NA	Yes	In 1 case, behavioral abnormalities since the age of 4	NA	NA	Prompting/pacing/shaping	+	≥14 years
Charlot	2002	11	5/6	22.7	Slowness, remain motionless, mutism, tics, OCS, further neurological signs, depression	Yes	NA	Yes	Yes	Down's syndrome	NA	NA	SSRI, clorpromazine ^a	+	NA
Singh	2003	1	M	15	Slowness, OCS, mannerisms, incontinence	NA	NA	Yes	Yes	Normal	Laboratory: hypothyroidism, EEG-normal	CT-normal	Exposure/response prevention	+	9 months
Lam	2008	1	M	NA	Slowness, reduced verbal responses	NA	Yes	NA	Yes	Intellectual disability	NA	NA	Pacing/prompting	±	9 weeks
Mittal	2013	1	M	12	Slowness, mutism, OCS, anxiety, depression	Yes	No	NA	Yes	NA	NA	NA	Modeling/prompting/pacing	+	24 weeks

^aExact gender details not given for all presented cases.^bData presented for only 2 cases.^cFour shared cases.

M/F, male/female; Tx, treatment; NA, not available; OCS/OCD, obsessive-compulsive symptoms/disorder; TCA, tricyclic antidepressants; BZP, benzodiazepines; OFC/OMC/PMC, orbitofrontal/mid-frontal/premotor cortex; SSRI, selective serotonin reuptake inhibitor.

neurological abnormalities in a large proportion of this cohort.² Subsequently, Berthier et al. reported on 3 patients diagnosed with OS with brain lesions, who were slow owing to overt obsessive-compulsive rituals after traumatic brain injury.¹⁶ The heterogeneity of terminology and diversity of applied diagnostic criteria and associated conditions has fueled the debate as to whether OS denotes a distinct and primary condition at all.⁴

Our 3 cases previously diagnosed with OS highlighted here were characterized by progressive motor slowness with poverty of movement. Fatiguing and decrement of movement during action repetition, cardinal features of parkinsonism and a marker of nigrostriatal degeneration,¹⁷ were not observed clinically, an observation supported by the normal dopamine transporter single-photon emission computerized tomography and [¹⁸F]dopa-PET scans of cases 1 and 2, respectively. Furthermore, all 3 patients were characterized by a diminution in speech strength, fluency, and prosody. Bizarre postures and mannerisms were prominent clinical features. Echophenomena and oculogyric tics were also noted in case 1. Interestingly, there was normalization in motor speed for reflexive, automated, and/or less attention-demanding movements, such as reflexive saccadic eye movements, or motor responses to unexpected environmental stimuli. In cases 1 and 2, there was also a short lasting normalization of motor behavior during arousal states (vegetative or emotional). Extreme distractibility was present in case 1. Obsessive-compulsive symptoms were present in all cases and autistic features in one of them (case 3). There were no psychotic symptoms.

Clearly, many of the aforementioned features of the cases described here, such as profound difficulties in initiating voluntary action, slowness of movement, poor speech production, motor perseverations, and abnormal posturing, could also be positioned within the phenomenological spectrum of catatonia. In fact, together with echophenomena (case 1), most constitute current diagnostic criteria for catatonia (Table 2).¹⁸ In addition, the short-lived episodes of vigorous and emotionally fueled motor responses have been described in this context as catatonic

excitement.¹⁹ Similarly, many cases described in the literature^{2,7,8,10,13} may also fall under the same diagnostic rubric, given that there is a clear overlap between what has been described as OS and the clinical syndrome of catatonia. Also, some of the neurological conditions associated with the manifestation of OS in the literature, such as Down's syndrome and autistic spectrum disorder, are also associated with catatonic symptoms.^{7,20–22} The hypothesis that the two might, in fact, constitute a spectrum with OS representing a *forme fruste* of catatonia has already been suggested.²³

Perhaps one source of confusion in the literature to date has been the use of the word “primary” in the original report of OS.¹ This is usually used to denote a separate, discrete disease entity, but reading the original report, this may not have been the researchers' intention. We interpret the use of this term in the original case series as an attempt to separate the phenomenon of extreme motor slowness in patients with OS, from common overt obsessive-compulsive behaviors that would be expected to cause motor slowness (e.g., checking rituals). The patients described did clearly have OCD, but their slowness was a peculiar and very disabling symptom seemingly unrelated to overt obsessive-compulsive rituals. Characterizing OS as an unusual symptom that can occur in OCD, rather than as a primary disease entity in its own right, may help make sense of later reports of OS in patients with OCD and neurological disorders known to be associated with OCD, for example, GTS, autistic spectrum disorder, and Down's syndrome.^{2,7,8,10,13} Furthermore, the recognition that catatonia is, in fact, a phenomenon that occurs outside the setting of schizophrenia,²⁴ for example, in OCD,²³ provides an explanation for the overlap between symptoms observed in patients with OS and the diagnostic criteria for catatonia. On one side of the phenomenological spectrum, patients with obsessive-compulsive symptoms might be diagnosed with OS, where the motor symptoms are likely to be caused by ruminations, covert rituals, compulsive repetition of action or their monitoring and hence action interruption, and avoidance strategies.^{3,4} On the other end of the spectrum, patients with obsessive-compulsive symptoms, not necessarily OCD, could present with aforementioned motor signs, but also additional symptoms, including the ones presented in our 3 cases here reaching diagnostic threshold for catatonia.

There are, however, also other neuropsychiatric causes in the absence of medication or intoxication that may present with severe motor slowness and belong in the differential diagnosis of OS. Psychomotor slowing is characteristic, for example, in major depression.^{25–27} Movement execution is impaired both for internally and stimulus-driven motor output,²⁸ but to a lesser extent than in the cases with OS presented here, and particularly case 1. However, OCD and depression often co-occur, and therefore in severe cases, it might be difficult to disentangle the separate contribution of the two disorders in manifestation of psychomotor slowness. Although none of our 3 patients had depression, this diagnostic difficulty is highlighted by the misdiagnosis of our first patient with depression early on during his illness, but also indeed by the presence of depressive symptoms

TABLE 2 DSM-5 criteria for diagnosis of catatonia

Stupor (i.e., no psychomotor activity; not actively relating to environment)
Catalepsy (i.e., passive induction of a posture held against gravity)
Waxy flexibility (i.e., slight and even resistance to positioning by examiner)
Mutism (i.e., no, or very little, verbal response)
Agitation, not influenced by external stimuli
Negativism (i.e., opposing or not responding to instructions or external stimuli)
Posturing (i.e., spontaneous and active maintenance of a posture against gravity)
Mannerisms (i.e., odd, circumstantial caricature of normal actions)
Stereotypies (i.e., repetitive, abnormally frequent, non-goal-directed movements)
Grimacing
Echolalia (i.e., mimicking another's speech)
Echopraxia (i.e., mimicking another's movements)

The presence of three or more criteria warrants the diagnosis of catatonia. Adapted from *Diagnostic and Statistical Manual of Mental Health Disorders*, Fifth Edition (DSM-5)¹⁸.

in approximately half of the identified cases in the literature review.^{2,7,10,12,14} Another differential that merits attention is apathy. Although apathy can be linked to depression, it represents a distinct syndrome.²⁹ Apathy related to disruption of "autoactivation" processing (also termed "psychic akinesia" or "athymhormia") is characterized by a reduction of voluntary motor output for self-generated movements paralleled by preservation of (short-lived) externally driven responses.²⁹ It is commonly related with lesions of the frontomesial cortex or the internal segment of the globus pallidus.²⁹ However, all our cases were characterized by overwhelming slowness of movement (both internally and externally driven), rather than simply a reduction in the frequency of self-generated movement itself. Finally, functional (psychogenic) parkinsonism is also a relevant consideration. Although motor slowness is the hallmark feature in these patients, asymmetric presentation, tremulousness, the presence of additional functional neurological signs, and the absence of obsessive-compulsive behavior facilitate diagnostic distinction.^{30–32}

To conclude, we propose that OS is a rare, but often disabling, motor manifestation of OCD, which is not associated with overt obsessive-compulsive behaviors that would be expected to lead to motor slowness. Some of these patients may also be diagnosed as catatonic, but the underlying pathophysiology appears to be related to OCD. This understanding could influence treatment selection in these patients, favoring the trial of established treatments for OCD. However, we recognize that some cases really do sit on the edge of current diagnostic criteria and further study will hopefully help define this disabling condition more precisely.

Author Roles

(1) Research Project: A. Conception, B. Organization, C. Execution; (2) Statistical Analysis: A. Design, B. Execution, C. Review and Critique; (3) Manuscript: A. Writing of the First Draft, B. Review and Critique.

C.G.: 1A, 1B, 1C, 3A, 3B

P.K.: 1A, 1B, 1C, 3A, 3B

M.C.: 1A, 1B, 1C, 3A, 3B

R.E.: 1C, 3A, 3B

B.B.: 1C, 3A, 3B

G.P.: 1C, 3A, 3B

M.J.E.: 1A, 1B, 1C, 3A, 3B

K.P.B.: 1A, 1B, 1C, 3A, 3B

A: Drafting/revising the manuscript for content, including medical writing for content. B: Acquisition of data. C: Study supervision or coordination. CG, PK, MC, MJE, KPB: A,B,C. RE, BB, GP: AB.

Disclosures

Funding Sources and Conflicts of Interest: The authors report no sources of funding and no conflicts of interest.

Financial Disclosures for previous 12 months: C.G. receives academic research support by the Deutsche Forschungsgemeinschaft (DFG; GA2031/1) and received commercial

research support in the form of travel grants by the International Parkinson and Movement Disorder Society (MDS), Actelion, Ipsen, Pharm Allergan, and Merz Pharmaceuticals. P.K. receives funding by The Bachmann-Strauss Dystonia & Parkinson Foundation. R.E. has been partly supported by COST Action BM1101 (reference: ECOST-STSM-BM1101-160913-035934). B.B. has received a research grant from the Gossweiler Foundation and travel grants from the MDS and the EFNS-ENS. M.J.E. receives royalties from Oxford University Press; receives research support from a National Institute for Health Research grant for a study in which he is the principal investigator and from Parkinson's UK, UK Dystonia Society, and the Guarantors of Brain; and has received honoraria for speaking from UCB. K.P.B. has received funding for travel from GlaxoSmithKline (GSK), Orion Corporation, Ipsen, and Merz Pharmaceuticals, LLC; serves on the editorial boards of *Movement Disorders* and *Therapeutic Advances in Neurological Disorders*; receives royalties from Oxford University Press; received speaker honoraria from GSK, Ipsen, Merz Pharmaceuticals, LLC, and Sun Pharmaceutical Industries Ltd.; received personal compensation for serving on the scientific advisory boards of GSK and Boehringer Ingelheim; received research support from Ipsen and from the Halley Stewart Trust through Dystonia Society UK, and the Wellcome Trust MRC strategic neurodegenerative disease initiative award (ref. no.: WT089698), as well as a grant from the Dystonia Coalition and a grant from Parkinson's UK (ref. no.: G-1009).

References

1. Rachman S. Primary obsessional slowness. *Behav Res Ther* 1974;12:9–18.
2. Hymas N, Lees A, Bolton D, Epps K, Head D. The neurology of obsessional slowness. *Brain* 1991;114:2203–2233.
3. Ratnasuriya RH, Marks IM, Forshaw DM, Hymas NF. Obsessive slowness revisited. *Br J Psychiatry* 1991;159:273–274.
4. Veale D. Classification and treatment of obsessional slowness. *Br J Psychiatry* 1993;162:198–203.
5. Bennun I. Obsessional slowness: a replication and extension. *Behav Res Ther* 1980;18:595–598.
6. Bilsbury C, Morley S. Obsessional slowness: a meticulous replication. *Behav Res Ther* 1979;17:405–408.
7. Charlot L, Fox S, Friedlander R. Obsessional slowness in Down's syndrome. *J Intellect Disabil Res* 2002;46:517–524.
8. Clark DA, Sugrim I, Bolton D. Primary obsessional slowness: a nursing treatment programme with a 13-year-old male adolescent. *Behav Res Ther* 1982;20:289–292.
9. Lam W, Wong KW, Fulks MA, Holsti L. Obsessional slowness: a case study. *Can J Occup Ther* 2008;75:249–254.
10. Mittal AK, Majumder P, Agrawal A, Sood M, Khandelwal SK. Early onset obsessive compulsive disorder with obsessive slowness: a case report and demonstration of management. *Indian J Psychol Med* 2013;35:407–409.
11. Sawle GV, Hymas NF, Lees AJ, Frackowiak RS. Obsessional slowness. Functional studies with positron emission tomography. *Brain* 1991;114:2191–2202.
12. Sawle GV, Lees AJ, Hymas NF, Brooks DJ, Frackowiak RS. The metabolic effects of limbic leucotomy in Gilles de la Tourette syndrome. *J Neurol Neurosurg Psychiatry* 1993;56:1016–1019.
13. Singh G, Sharan P, Grover S. Obsessive slowness : a case report. *Indian J Psychiatry* 2003;45:60–61.
14. Takeuchi T, Nakagawa A, Harai H, et al. Primary obsessional slowness: long-term findings. *Behav Res Ther* 1997;35:445–449.

15. Tham SW, Sarathchandra CB. Obsessional slowness in the setting of fears of contamination resulting in paradoxical uncleanliness. *Br J Hosp Med* 1994;51:67–68.
16. Berthier ML, Kulisevsky J, Gironell A, Heras JA. Obsessive-compulsive disorder associated with brain lesions: clinical phenomenology, cognitive function, and anatomic correlates. *Neurology* 1996;47:353–361.
17. Hughes AJ, Daniel SE, Kilford L, Lees AJ. Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinico-pathological study of 100 cases. *J Neurol Neurosurg Psychiatry* 1992;55:181–184.
18. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Health Disorders: DSM-5*. Washington, DC: American Psychiatric Publishing; 2013.
19. Gjessing LR. A review of periodic catatonia. *Biol Psychiatry* 1974;8:23–45.
20. Dhossche D. Brief report: catatonia in autistic disorders. *J Autism Dev Disord* 1998;28:329–331.
21. Jap SN, Ghaziuddin N. Catatonia among adolescents with Down syndrome: a review and 2 case reports. *JECT* 2011;27:334–337.
22. Wing L, Shah A. Catatonia in autistic spectrum disorders. *Br J Psychiatry* 2000;176:357–362.
23. Fontenelle LF, Lauterbach EC, Telles LL, Versiani M, Porto FH, Mendlowicz MV. Catatonia in obsessive-compulsive disorder: etio-pathogenesis, differential diagnosis, and clinical management. *Cogn Behav Neurol* 2007;20:21–24.
24. Taylor MA, Fink M. Catatonia in psychiatric classification: a home of its own. *Am J Psychiatry* 2003;160:1233–1241.
25. Lohr JB, May T, Caligiuri MP. Quantitative assessment of motor abnormalities in untreated patients with major depressive disorder. *J Affect Disord* 2013;146:84–90.
26. Rogers D, Lees AJ, Smith E, Trimble M, Stern GM. Bradyphrenia in Parkinson's disease and psychomotor retardation in depressive illness. An experimental study. *Brain* 1987;110:761–776.
27. Sabbe B, Hulstijn W, Van Hoof J, Zitman F. Fine motor retardation and depression. *J Psychiatr Res* 1996;30:295–306.
28. Hoffstaedter F, Sarlon J, Grefkes C, Eickhoff SB. Internally vs. externally triggered movements in patients with major depression. *Behav Brain Res* 2012;228:125–132.
29. Levy R, Dubois B. Apathy and the functional anatomy of the prefrontal cortex-basal ganglia circuits. *Cereb Cortex* 2006;16:916–928.
30. Benaderette S, Zanotti Fregonara P, Apartis E, et al. Psychogenic parkinsonism: a combination of clinical, electrophysiological, and [(123)I]-FP-CIT SPECT scan explorations improves diagnostic accuracy. *Mov Disord* 2006;21:310–317.
31. Jankovic J. Diagnosis and treatment of psychogenic parkinsonism. *J Neurol Neurosurg Psychiatry* 2011;82:1300–1303.
32. Lang AE, Koller WC, Fahn S. Psychogenic parkinsonism. *Arch Neurol* 1995;52:802–810.

Supporting Information

Videos accompanying this article are available in the supporting information here.

Video 1. Clinical examination of case 1. Segment A: Facial impassivity with normal blink rate, repetitive perioral movements, poverty of movement with prolonged movement initiation, and slowed execution. Segment B: Oculogyric tics. Segment C: Profound slowing during arm elevation and finger tapping. Slow hesitant gait with small steps. Segment D: Passive induction of postures held against gravity in the absence of any verbal instruction (waxy flexibility).

Video 2. Clinical examination of case 2. Careful, manneristic movements. Normal finger tapping. Meticulous and slow movement execution. Slow hesitant gait with bilateral dorsal shoulder flexion.

Video 3. Gait examination of case 3. Slow hesitant gait with bilateral dorsal flexion and abduction of shoulders.